

MMPI-2-RF AND EEG CORRELATES OF DISORDERED EATING BEHAVIORS IN A
CLINICAL AND NON-CLINICAL SAMPLE

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ABSTRACT

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Eating disorders produce the highest mortality rates of any mental health diagnoses; between 5-20% of individuals struggling with anorexia nervosa will die from it or associated medical complications. In an expansive study of adult women with anorexia nervosa, Steinhausen (2002) found that less than half of individuals with anorexia had a full recovery, one third of individuals improved, and 20.8% of individuals lived with chronic anorexia. In a study of women with bulimia nervosa, Steinhausen (2009) found the overall mortality rate to be between .3 and 3.1%. In surviving patients, slightly less than half of individuals with bulimia had a full recovery, 26% of individuals improved, and 26% of individuals lived with chronic bulimia. Binge-eating disorder appears to be relatively persistent and the course is comparable to that of bulimia nervosa in terms of severity and duration (American Psychiatric Association, 2013).

Additionally, there is also a high prevalence of comorbid psychiatric and personality disorders associated with eating disorders. Based on a national comorbidity survey, 56.2% of respondents with anorexia nervosa, 94.5% of respondents with bulimia nervosa, and 78.9% of respondents with binge-eating disorder qualified for at least one core mood, anxiety, or substance use disorder in the DSM-IV (Hudson et al., 2012). Considering these rates of comorbidity, research

advances in the relationship between psychopathology and eating disorders could increase the diagnostic specificity and accuracy of eating disorders. Along with comorbidity, research also suggests that there are physiological markers associated with eating disorders, including abnormal brain wave activity. Hatch et al. (2011) found that individuals with anorexia have EEG abnormalities both before and after refeeding, specifically low alpha waves and increased beta and theta waves. Greenblatt et al. (2011) suggested that EEG data could assist clinicians in identifying individuals suffering from eating disorders with associated depressive and anxiety symptoms. Results suggested that patients whose treatment modalities were guided by Key EEG data reported significant decreases in depressive symptoms and severity of eating disorder and a significant increase in overall clinical improvement. These results are encouraging due to eating disorders being difficult to treat. This study examined the relationship between personality and psychopathology, as identified by MMPI-2-RF scales, EEG activity, and disordered eating in a clinical and non-clinical sample of adult women. The non-clinical sample included individuals with mild or no disordered eating to provide a normal comparison group. The clinical sample included women diagnosed with a recognized eating disorder. Specific measures of disordered eating behavior were collected. Results may identify additional EEG markers or personality indicators that are associated with disordered eating. Additional data about psychopathologies that are comorbid with disordered eating could advance treatment specificity and efficacy. The research functioned under the Research Domain Criteria (RDoC) framework to investigate relationships between psychological constructs and physiological measures. This paper will review the history and current diagnostic conceptualization of eating disorders, previous literature outlining the use of the MMPI and EEG correlates to distinguish disordered eating behaviors, and will outline the hypotheses, methodology, and results of this study.

CHAPTER 1: INTRODUCTION

Eating disorders are notable as the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) sector producing the highest mortality rates of any mental health diagnoses; between 5-20% of individuals struggling with anorexia nervosa will die from it or associated medical complications. Mortality statistics show that deaths associated with chronic eating disorders can result from heart failure, organ failure, gastric rupture, or suicide (National Eating Disorder Association, 2015). For those who survive, the disorder is often chronic and lengthy. In an expansive study of adult women with anorexia nervosa, Steinhausen (2002) found that less than half of individuals with anorexia had a full recovery, one third of individuals improved, and 20.8% of individuals lived with chronic anorexia. The long-term outcome of bulimia nervosa is only slightly better as compared to anorexia nervosa; however, the rate of mortality is lower. In an expansive study of women with bulimia nervosa, Steinhausen (2009) found the overall mortality rate to be between .3 and 3.1%. In surviving patients, slightly less than half of individuals with bulimia had a full recovery, 26% of the individuals improved, and 26% of individuals lived with chronic bulimia. Remission rates in outcome studies are higher for binge-eating disorder than for bulimia nervosa or anorexia nervosa. Binge-eating eating disorder appears to be relatively persistent and the course is comparable to that of bulimia nervosa in terms of severity and duration (American Psychiatric Association, 2013). Prior to 2013, binge-eating disorder was included in the Eating Disorder Not Otherwise Specified diagnosis of the DSM-IV, along with other disordered eating behaviors (American Psychiatric Association, 1994). Due to its short history as an independent diagnosis, recovery and mortality statistics for binge-eating disorder are inconsistent; however, the mortality rate for Eating Disorder Not

Otherwise Specified was previously reported as 5.2% (Crow et al., 2009). One outcome study found an estimated mortality rate of 2.9% for binge-eating disorder independently (Smink, Hoeken & Hoek, 2012).

Current lifetime prevalence of anorexia nervosa, bulimia nervosa, and binge eating disorder among females are .9%, 1.5%, and 3.5% respectively (Hudson, Hiripi, Pope & Kessler, 2012). There is also a high prevalence of comorbid psychiatric and personality disorders associated with eating disorders. Based on a national comorbidity survey, 56.2% of respondents with anorexia nervosa, 94.5% of respondents with bulimia nervosa, and 78.9% of respondents with binge eating disorder qualified for at least one core mood, anxiety, impulse-control, or substance use disorder in the DSM-IV (Hudson et al., 2012). Considering these rates of comorbidity, research advances in the relationship between psychopathology and eating disorders could increase the diagnostic specificity and accuracy of eating disorders.

Along with co-morbidity, research also suggests that there are physiological markers associated with eating disorders, including abnormal brain wave activity. Hatch et al., (2011), found that individuals with anorexia have electroencephalography (EEG) abnormalities both before and after refeeding, specifically low alpha waves and increased beta and theta waves. Therefore, identifying EEG patterns that correlate with eating disorders could be a valuable diagnostic tool. Greenblatt et al., (2011) suggested that EEG data could assist clinicians in identifying individuals suffering from eating disorders with associated depressive and anxiety symptoms. Results suggested that patients whose treatment modalities were guided by key EEG data (reference EEG or rEEG) reported significant decreases in depressive symptoms and severity of eating disorder and a significant increase in overall clinical improvement. These results are encouraging due to eating disorders being difficult to treat.

This study examined the relationship between personality and psychopathology, as identified by the Minnesota Multiphasic Personality Inventory-2-Restructured Form (MMPI-2-RF) scales, EEG activity, and disordered eating in a clinical and non-clinical sample of adult women. The non-clinical sample included individuals with mild or no disordered eating to provide a normal comparison group. The clinical sample included women diagnosed with a DSM-V recognized eating disorder. Specific measures of disordered eating behavior were collected. The procedure used the diagnostic categories as inclusion criteria but these categories will not be part of the subsequent analyses. The intent of this study was to identify additional EEG markers or personality indicators that are associated with disordered eating. Additional data and even replication of previous findings about psychopathologies that are comorbid with disordered eating could advance treatment specificity and efficacy. The research functioned under the Research Domain Criteria (RDoC) framework to investigate relationships between psychological constructs and physiological measures. This paper will review the history and current diagnostic conceptualization of eating disorders, previous literature outlining the use of the MMPI and EEG correlates to distinguish disordered eating behaviors, and will outline the hypotheses, methodology, and results of this study.

CHAPTER 2: LITERATURE REVIEW

History of Eating Disorders

The first clinical documentation of an eating disorder, specifically anorexia nervosa type behavior, is found as early as the 1600's. An English physician, Richard Morton, described two cases of a "wasting disease of nervous origins" in *Phtisiologia: a Treatise on Consumption* published in 1689. Following Morton, other physicians described similar cases, such as Baglivi in the early 1700s, Robert Whytt in 1764 and Louis-Victor Marce in 1860 (Gordon, 2000). These cases lacked detail and were described without depth, but they document the long-term presence of anorexia nervosa in human history. Anorexia nervosa did not gain its name and distinct attention in the medical field until the middle of the nineteenth century. Initially, anorexia type behavior was classified under the umbrella term "hysteria." Sir William Gull, a physician to Queen Victoria, coined the term anorexia nervosa and distinguished the condition as a psychological disorder in his 1874 article (Hepworth, 1999). Considering the time period, Gull's clinical description is remarkably similar to the modern understanding of the disorder. In his article describing two patients, he notes their emaciation, lack of menstrual periods, slowed pulse and breathing, low temperature, motor restlessness, and compulsive exercise. He goes as far as to distinguish both of the current subtypes, suggesting bingeing by noting occasional periods of 'voracious appetite' with one of his patients. While Gull's peers were classifying anorexia nervosa as a form of hysteria or a gastro-intestinal issue, he was ahead of his time stating, "The importance of diagnosing such cases in practice is obvious; otherwise, prognosis will be erroneous and treatment misdirected" (Madden, 2004).

Little progress was made after Gull's initial publication. Even up to the 1930's anorexia nervosa had nearly disappeared as a clinical finding. In the early 1900's, there was a newly discovered endocrine disease that mimicked the physical weight loss of anorexia nervosa. This new disease may have convinced clinicians that Gull's psychological explanation was less credible (ANRED, 2008). In the late 1930's, the endocrine disease and anorexia nervosa were clearly differentiated. According to Madden (2004), researcher Gerald Russell suggested that abnormal body image, weight phobia, and fear of fatness were not consistently clinically noted until the 1960's by professionals such as Bruch, Crisp and Russell. By the mid-1980's, additional symptoms, such as laxative and diuretic abuse, excessive exercise, and purging by vomiting, were being documented in higher numbers and associated with the disorders (Ash & Piazza, 1994). Anorexia has also seen significant change of recognition in the DSM. In 1952, anorexia nervosa was placed in the DSM-I as a psycho-physiological reaction, which was considered a neurotic illness (American Psychiatric Association, 1952). In 1968, the DSM-II revised the placement and category of these disorders placing them under special symptoms-feeding disturbances, along with pica and rumination (American Psychiatric Association, 1968). Beginning in the DSM-III in 1980, the features of abnormal body image, weight phobia, and fear of fatness began appearing as essential features for diagnosis. In 1994, the DSM-IV further modified the diagnostic criteria to reflect empirical findings about the disorders. The DSM-IV also differentiated between the restriction type and binge/purge type. These types caused significant diagnostic change. For example, in the DSM-III-R an anorexia nervosa patient who engaged in Binge/purge behavior also received the bulimia diagnosis. The changes in the DSM-IV suggested that anorexia behavior in the two subtypes consistently functioned differently (Sunday et al., 2001).

Bulimia nervosa was first named and defined independent of anorexia nervosa, in 1979 by Gerald Russell in a published clinical paper (Steinhausen, 2009). Binge/purge behavior had long been observed in anorectic patients prior to the behavior being categorized as a separate disorder. The long history of seeing Binge/purge behavior in people diagnosed as anorectic suggests that bulimia nervosa may have been present long before 1979. At that time, Russell also noted personality traits he found to be associated with bulimia, such as extroversion, impulsivity, and sexual promiscuity (Gordon, 2000). The DSM-III was the first to list the disorder as simply 'bulimia' (American Psychiatric Association, 1980). The diagnostic criteria for bulimia was problematically broad. The diagnosis required binge-eating behavior, but did not specify other disordered compensatory behaviors. In the DSM-III-R, the diagnosis of "bulimia nervosa" was much closer to what Russell had previously outlined, and listed both binge-eating and compensatory behaviors (Palmer, 2004). According to DSM-III-R, an individual could have a dual diagnosis of anorexia nervosa and bulimia nervosa. The DSM-IV included the first separation of bulimia subtypes of purging and non-purging, and added a weight requirement to differentiate the diagnosis of anorexia purge-type from bulimia (American Psychiatric Association, 1994). The DSM-5 maintained all the bulimia criteria from the previous edition, but reduced the frequency of binge-eating and compensatory behaviors from twice a week to once a week (American Psychiatric Association, 2013).

The diagnosis of bulimia has increased steadily since Russell defined the disorder (Hudson et al., 2012). However, the diagnostic specifics have changed so dramatically since 1979 that exact prevalence increases cannot be estimated accurately.

Binge-eating disorder is recognized for the first time in the DSM-5 (American Psychiatric Association, 2013). However, recognition of non-compensatory, compulsive eating occurred far

before it received individualized diagnostic criteria. Albert Stunkard first introduced the concept of “overeating without compensatory behaviors” in 1959. It was conceptualized and structured as a specific syndrome in the 1990’s by Fairburn and Spitzer (Amianto, Ottone, Abbate & Fassino, 2015). Binge-eating disorder was first mentioned in the DSM-IV in 1994. It was listed in Appendix B as an eating disorder that needed further research, and it was placed under the criteria for Eating Disorder Not Otherwise Specified (American Psychiatric Association, 1994). The official diagnosis in the DSM-5 maintained diagnostic criteria consistent with its representation in the DSM-IV, but lowered frequency and duration of behaviors required for diagnosis (Amianto et al., 2015)

Review of Current Diagnostic Criteria

Anorexia Nervosa

Currently, the DSM-5 lists three criteria required for a diagnosis of anorexia nervosa:

- 1) a restriction of energy intake relative to requirements leading to a significantly low body weight,
- 2) an intense fear of gaining weight or becoming fat, or persistent behavior that interferes with weight gain, even though at a significantly low weight, and
- 3) a disturbance in the way in which one’s body weight or shape is experienced, undue influence of body weight or shape on self-evaluation, or persistent lack of recognition of the seriousness of the current low body-weight (American Psychiatric Association, 2013).

The DSM-5 also gives two specifiers for anorexia nervosa. In the restricting type, clients present with weight loss that is accomplished primarily through dieting, fasting, and/or excessive exercise with the absence of binge/purge behavior. In the binge-eating/purging type, a client has a minimum of three months of recurrent episodes of binge eating and/or purging behavior.

Specifically, purging is dictated as self-induced vomiting or the misuse of laxatives, diuretics, or enemas. Anorexia with purgative behavior provides unfavorable prognostic factors for recovery and mortality statistics (Steinhausen, 2009).

Individuals with anorexia often experience medical symptoms such as anemia, dehydration, sinus bradycardia, poor bone mass with increased risk of fracture, and significant reduction in resting energy expenditure. Many of the physical signs and symptoms are attributable to starvation, such as amenorrhea in females, constipation, lanugo, peripheral edema, and intolerance to cold (American Psychiatric Association, 2013). Only an estimated 33% of individuals with anorexia will receive treatment for the disorder (Eating Disorder Statistics & Research, 2016).

Bulimia Nervosa

The DSM-5 lists three criteria required for a diagnosis of bulimia nervosa:

- 1) recurrent episodes of binge eating, specifically with a sense of lack of control over eating, and
- 2) recurrent inappropriate compensatory behaviors in order to prevent weight gain, such as self-induced vomiting, misuse of laxatives, diuretics, fasting, or excessive exercise; both of these criteria must occur on average at least once a week for a three month duration, and
- 3) self-evaluation is unduly influenced by body shape and weight, and the disturbance does not occur exclusively during episodes of anorexia nervosa.

An episode of binge eating is defined as “eating, in a discrete period of time, an amount of food that is definitely larger than what most individuals would eat in a similar period of time under similar circumstances” (American Psychiatric Association, 2013, p. 345). These symptoms are often experienced with a sense of shame and active secrecy. Individuals of this disorder may closely resemble those with anorexia nervosa in the experiential symptoms including fear of

gaining weight, desire to lose weight, and intense dissatisfaction with their body shape. The most common antecedent of binge eating is negative affect (American Psychiatric Association, 2013). Individuals with bulimia are typically within the normal weight or overweight range (body mass index (BMI) ≥ 18.5 and < 30). Between binges, individuals with bulimia typically restrict their total caloric consumption and often select low calorie foods, while avoiding high calorie foods and foods typically consumed during a binge. These restriction periods vary in duration per case. Medical issues associated with bulimia include menstrual irregularity, electrolyte disturbances, gastrointestinal complications, metabolic acidosis, and extensive loss of dental enamel. More serious complications include esophageal tears, gastric rupture, rectal prolapse, and cardiac arrhythmias. Kessler et al. (2013) found that psychological issues due to bulimia include role impairment in 54.5% of respondents, and severe role impairment was in 21.8%. The prevalence of these effects could be underestimated due to the secretive nature of the disorder and a reliance on self-report measures in many studies. Only an estimated 6% of individuals with bulimia will receive treatment for the disorder (Eating Disorder Statistics & Research, 2016).

Binge-eating Disorder

The DSM-5 lists four criteria for diagnosis of binge-eating disorder:

- 1) recurrent episodes of binge eating as previously described, at least once a week for 3 months,
- 2) lack of compensatory behaviors for binge eating,
- 3) marked distress regarding binge eating, and
- 4) binge eating must be associated with at least three of the following features: eating more rapidly than normal, eating until feeling uncomfortably full, eating large amounts of food without physical hunger cues, eating alone due to embarrassment of eating behaviors, or feelings of disgust, depression or guilt after.

Some individuals report a dissociative quality during or after binge eating. As with bulimia nervosa, binge eating is associated with increased shame and secrecy (American Psychiatric Association, 2013). Binge-eating disorder typically occurs in normal-weight, overweight, and obese individuals, particularly the latter two in treatment-seeking individuals. For diagnostic purposes, severity with binge-eating disorder is linked to the intensity of the binge-eating and not the degree of obesity. It is important to note the psychological component of binge-eating disorder and its distinction from obesity. Most obese individuals do not engage in recurrent binge eating, do not experience the associated functional impairment, do not present greater psychiatric comorbidity, or experience significant subjective distress. All of these things are common individuals with binge-eating disorder. Overvaluation of shape and weight is present in 60% of individuals with binge-eating disorder. This is significantly higher than what is seen in non-diagnosed obese individuals (Amianto et al., 2015); however, it is significantly lower than the emphasis on shape and weight seen with anorexia and bulimia. Medical issues associated with binge-eating disorder are impaired health-related quality of life and life satisfaction, increased medical morbidity, and increased mortality. Kessler et al. (2013) found that role impairment due to binge-eating disorder was reported by 46.7% of respondents, and severe role impairment was reported by 13.2% of respondents. As with bulimia, the prevalence of these effects could be underestimated due to the secretive nature of the disorder and a reliance on self-report measures in many studies. An estimated 43% of individuals with binge-eating disorder will receive treatment for the disorder (Eating Disorder Statistics & Research, 2016).

Unspecified and Other Specified Feeding or Eating Disorders

The DSM-5 (American Psychological Association, 2013) also provides the diagnoses of Unspecified Feeding or Eating Disorders and Other Specified Feeding and Eating Disorders.

This category applies to when symptoms characteristic of an eating disorder that cause clinically significant distress or impairment in social, occupational or other important areas of functioning predominate, but the disorder does not meet the full criteria for the disorders described above. Criteria for duration, frequency, and/or weight for unspecified eating disorders are not defined.

The course and outcome of eating disorders are noted to have a degree of crossover from one diagnosis to the other. The prevalence of crossover in eating disorders is debated and statistics have been mixed (Tozzi et al., 2005); however, many studies have found crossover to be a frequent occurrence, especially for individuals initially diagnosed with anorexia. Milos et al. (2005) found over a 30-month time period only a third of individuals maintained their original eating disorder diagnosis. Eddy et al. (2008) found that over a seven-year time period nearly one third of individuals with anorexia crossed over to bulimia at some point. Also, 62% of individuals with anorexia crossed between the restricting type and binge/purge type of anorexia, indicating a clear change in disordered eating behavior. Diagnostic crossover from an initial diagnosis of bulimia nervosa to anorexia nervosa is less common and occurs in only 10-15% of cases. These individuals who experience crossover from an initial diagnosis of bulimia will commonly revert back to bulimia or have multiple crossovers between bulimia and anorexia. An unidentified subset of individuals with bulimia nervosa continue to binge eat but no longer engage in inappropriate compensatory behaviors, and therefore crossover to a binge-eating disorder diagnosis (American Psychiatric Association, 2013). The majority of eating disorder diagnosis crossovers will occur in the first five years of the disorder. At this time clinicians are limited in their ability to predict who is likely to experience diagnostic crossover (Tozzi et al., 2005); however, expanding research that is behavior-specific could clarify this distinction.

For example, the primary diagnostic difference between purge-type anorexia and bulimia hinges only on body weight. Thus the transition between the two diagnoses could effectively be a change in stage of illness, rather than an actual change in behavior. The significant symptom overlap and tendency to be diagnosed with multiple eating disorder diagnoses over the course of an individual's life suggests a need to reconsider the diagnostic criteria for eating disorders. Diagnoses should avoid using arbitrary diagnostic cut offs that yield nonspecific and convoluted research results. Emphasizing specific behaviors rather than rigid boundaries and categories may produce more clinically relevant results and offer more accurate diagnostic measures.

Research Domain Criteria

The National Institute of Mental Health's (NIMH) Research Domain Criteria (RDoC) project focuses on a research framework that redirects research in psychopathology to focus on multi-level specific, measurable constructs. The project integrates many levels of information to include sources of data that provide correlations between psychological data and physiological data to better understand basic dimensions of psychopathology functioning without arbitrary diagnosis cutoffs ("Research Domain Criteria," n.d.). The RDoC project is working to shift thinking about mental health dysfunction from a categorical diagnostic framework into a dimensional framework of psychopathology with neurobiological ties.

The RDoC project is calling for more research investigating the relationship between brain imaging and eating disorders stating, "Brain imaging studies are also providing a better understanding of eating disorders.... This kind of research can help guide the development of new means of diagnosis and treatment of eating disorders." ("Research Domain Criteria," n.d., para 8)

The structure of this study is in line with the RDoC framework by collecting dimensional, non-categorical symptom data, neurobiological physiological measures of brain wave activity, and MMPI-2-RF data that reasonably measure the full domain of psychopathological traits in a dimensional fashion.

Psychopathology, Personality, and Disordered Eating

Research has shown that a considerable number of individuals who are diagnosed with an eating disorder meet diagnostic criteria for an additional psychopathology, predominantly anxiety, depressive, and personality disorders. Hatch et al. (2012) found that more than half of individuals with anorexia, bulimia, and binge-eating disorder will receive treatment for non-eating related emotional problems at some point in their lives. The cause for the high levels of comorbidity among eating disorders is unknown; however, there is evidence that common genetic factors may contribute to the co-occurrence of eating disorders and mood disorders (Hatch et al., 2012).

Steinhausen (2009) found that anorexia has a high level of comorbidity that spans DSM-IV categories, including neurotic and anxiety disorders (25.5%), obsessive compulsive disorders (12%), schizophrenia (4.6%), borderline or unspecified personality disorders (17.4%), and substance use disorders (14.6%). The study specifically noted that obsessive-compulsive behaviors add to chronicity for anorexia. Comorbid depression and anxiety in eating disorders often worsen due to malnutrition (Mattar, Thiebaud, Huas, Cebula, & Godart, 2012). Steinhausen (2002) also found that depression, anxiety, phobias, and personality disorders contribute to a worse prognosis for anorexia.

In an expansive study of eating disorder comorbidity, Kessler et al. (2013) found the vast majority (84.8%) of respondents with bulimia met lifetime criteria for other DSM-IV or

Composite International Diagnostic Interview (CIDI) disorders. Steinhausen (2009) also found that bulimia has a high level of comorbidity that spans DSM-IV categories, including affective disorders (25%), neurotic and anxiety disorders (5%), personality disorders (7%), and substance use disorders (10%). The DSM-5 also notes comorbidity with depressive disorders and bipolar disorder, both contributing to a negative prognosis. Additionally, approximately 30% of individuals with bulimia also meet the diagnostic criteria for a substance use disorder, most commonly stimulants or alcohol (American Psychiatric Association, 2013). Substance use comorbidity is higher in bulimia than in either of the other primary eating disorders. Cassin and Von Ranson (2005) found that patients diagnosed with both bulimia and borderline personality disorder showed clinically significant eating disorder symptoms over a longer period than patients diagnosed with just bulimia.

Additionally, in Kessler et al.'s (2013) comorbidity study, the vast majority (79%) of respondents with binge-eating disorder also met lifetime criteria for other DSM-IV/CIDI disorders. Personality, mood, and substance disorders are frequent comorbidities, and all relate to poor prognosis. These correlations are so prominent in outcome that they have been proposed as markers of severity, rather than just associated disorders (Amiato et al., 2015). Grilo, White, and Masheb (2009), found that 43.1% of their binge-eating disorder participants had at least one current, co-occurring psychiatric disorder. In terms of current comorbidity, mood disorders (26.0%) and anxiety disorders (24.5%) were most common. The study also found that they met lifetime criteria for mood disorders (54.2%), anxiety disorders (37.1%), and substance use disorders (24.8%). Additionally, the study also found that patients with current comorbidity had significantly higher levels of eating disorder behavior, negative affect, and lower self-esteem relative to patients with either past psychiatric history or no psychiatric histories. The DSM-5

highlights comorbidity with bipolar, depressive, anxiety, and substance abuse disorders (American Psychiatric Association, 2013).

Research suggests that eating disorders may also be correlated with personality differences and these personality differences may be predictive of symptomatology, treatment response, and prognosis. De Bolle et al. (2011) found that internalizing and externalizing factors of personality have an influence on symptomology and maintenance of symptoms in individuals with comorbid personality disorders and eating disorders. Global role functioning for individuals diagnosed with anorexia and bulimia was found to be significantly associated with the comorbid dimensions of both internalizing and externalizing personality pathology compared with individuals without comorbidity.

Perfectionism, obsessive compulsiveness, sensation seeking, and impulsivity are associated with both bulimia and anorexia. Besides symptoms that were characteristic of both disorders, analysis also revealed many personality differences between eating disorder types. Individuals with anorexia were characterized by high constraint, persistence and low novelty seeking, and individuals with bulimia were characterized by high impulsivity, sensation seeking, novelty seeking and traits often associated with borderline personality disorder (Cassin & Von Ranson, 2005). Perfectionism is a central feature of eating disorders, which may predate the onset of the disorder and persist after recovery. Obsessive-compulsive tendencies are frequently comorbid with eating disorders, but decrease in prevalence when binge/purge behavior is present (Halmi et al., 2005; American Psychiatric Association, 2015). In contrast, individuals with binge-eating behavior, purging behavior, or both tend to score higher on measures of sensation seeking than individuals with restricting type behaviors. These characteristic tendencies are seen as a need for control, perfectionist tendencies, and rigid thinking in anorexia and impulsivity,

sensation seeking, and substance abuse in bulimia (Cassin & Von Ranson, 2005; Abbate-Daga, Gramaglia, Malfi, Piero, & Fassino, 2007). Studies examining personality variables for binge-eating disorder suggest that they are more similar to bulimia than restricting-type anorexia. It may be that these traits affect the symptomatic expression of eating disorders, with high constraint and persistence predisposing to dietary restriction, and high impulsivity, sensation seeking and novelty seeking creating a vulnerability for bingeing and purging behaviors (Cassin & Von Ranson, 2005).

Previous research supports the association of consistent identifiable personality traits with the expression of specific disordered eating behaviors. Studies examining the association between personality and disordered eating symptomatology have proliferated in recent research, but there is still much to be explored; for example, few studies have been conducted on personality in binge-eating disorder. Most studies previously looking at personality and disordered eating behavior have done so with DSM categorical diagnoses (De Bolle et al., 2010). This approach reduces the chance to identify personality attributes with specific eating behaviors. Research using a dimensionalized approach to measuring both disordered eating behaviors and personality and psychopathology could systematically identify relationships between personality differences and disordered eating. This could create a diagnostic approach in which identifiable personality attributes, including psychopathology tendencies, allow an earlier diagnosis and more effective intervention for individuals with disordered eating.

Review of MMPI History and Disordered Eating

The Minnesota Multiphasic Personality Inventory has been one of the leading instruments for assessing personality and psychopathology since its creation in 1943. There have been ongoing demonstrations of its ability to measure clinically relevant characteristics of

personality and psychopathology in a variety of settings and populations. It measures relevant constructs in a dimensional fashion, and after extensive research it can be assumed to effectively measure the span of psychopathology and personality.

Development of the MMPI

The Minnesota Multiphasic Personality Inventory (MMPI) was first published in 1943 by authors Starke Hathaway and J. Charnley McKinley at the University of Minnesota Hospital for the purpose of efficient and effective routine diagnostic assessments. Hathaway and McKinley constructed the basic Clinical scales of the MMPI by first selecting 504 personality-type statements that they judged to be reasonably independent of each other. Secondly, they selected two criterion groups. The first group, the “Minnesota normals”, consisted primarily of 724 relatives and visitors of patients of the University of Minnesota Hospitals with no identified psychopathology. The second group, the clinical participants, included 221 patients, representing all of the major psychiatric categories being used clinically at that time, divided into clinical subgroups including hypochondriasis, depression, hysteria, psychopathic deviate, paranoia, psychasthenia, schizophrenia, and hypomania. Thirdly, they administered the 504 items to the two criterion groups, and through item analysis for each clinical subgroup, they determined which of the 504 items significantly differentiated between the clinical subgroup and the non-clinical criterion group. This resulted in a Clinical scale representing each psychiatric subgroup. Some additional changes were made to several scales to ensure the scales had discriminant ability. In 1946, Drake developed Social Introversion, the final Clinical scale, through item contrast on the introversion/extroversion scale of the Minnesota T-S-E Inventory (Evans & McConnell, 1941). Scores on each scale were represented as T scores with a mean of 50; scores above 70 on the MMPI were considered in the clinical range. Hathaway and

McKinley also created four validity scales in order to identify deviant test-takers and create an exclusion measure for falsified self-report results (Graham, 2011).

The construction of the test resulted in 10 primary scales. Scale 1, Hypochondriasis, was developed to identify patients who displayed symptoms associated with the diagnosis of hypochondriasis. High scores on Scale 1 were characteristic of preoccupation with the body and persistent fears of illness and disease. All item level content dealt with somatic concern or general physical competence. Scale 2, Depression, was developed to assess symptomatic depression. High scores on Scale 2 were characteristic of anhedonia, lack of energy, and denial of happiness. Scale 3, Hysteria, was developed to identify patients who were having hysterical reactions to stress situations. High scores on Scale 3 were characteristic of individuals who often feel overwhelmed, may develop physical symptoms as a reaction to turmoil, and often a lack of insight concerning the psychological underlying cause of physical symptoms. Scale 4, Psychopathic Deviate, was developed to identify patients who display delinquent behavior to a diagnostic level. High scores on Scale 4 likely suggest behaviors such as lying, stealing, sexual promiscuity, substance use, and difficulties with family and authority. Scale 5, Masculinity-Femininity, was originally constructed to diagnose homosexuality in men, which was considered a mental disorder at the time of the test creation. High T scores were associated with deviation from one's own sex. Scale 6, Paranoia, was created to identify patients with paranoid symptoms, such as suspiciousness, feelings of persecution, grandiose self concepts, and rigid opinions. High scorers in Scale 6 may appear psychotic, displaying thought dysfunction. Scale 7, Psychasthenia, was created to identify individuals with excessive doubts, compulsions and obsessions. This scale was a reliable index of psychological turmoil, characterized by agitation, anxiety and excessive worry. Scale 8, Schizophrenia, was developed to identify patients

diagnosed with schizophrenia. High scores would be characterized by behavior such as confusion, disorganization and disorientation, and unusual thought patterns. Scale 9, Hypomania, was developed to identify patients with hypomanic symptoms, characterized by elevated mood, accelerated speech and motor activity, and brief periods of depression. Scale 0, Social Introversion, was added later than the other clinical scales and was built to measure an individual's tendency to withdraw from social contacts and responsibility (Graham, 2011).

MMPI and Disordered Eating

The MMPI has been used in many studies to explore differences in scale elevations between eating disorder diagnoses and subtypes, primarily with anorexia and bulimia. For the purpose of this review, anorexia will be denoted as AN (restricting subtype (ANR) and purging subtype (ANP)), and bulimia as BN (purging type (BNP) and non-purging type (BNNP)). Researchers were looking for a consistent differentiation between the types reflected in differences in single elevations, 2-point code elevations, and 3-point code elevations.

Norman and Herzog (1983) compared MMPI profiles of patients diagnosed with ANR, ANP, and BN. They found an overall similarity between the MMPI configuration for each of the three groups, though certain scale differences characterized each group. Scales elevated to a clinical level could distinguish the three groups. ANR had only scale 2 clinically elevated, while BN had scales 2, 4, and 8, and ANP had seven scales elevated including 1, 2, 3, 4, 6, 7, and 8. This supports the previous literature that binge/purge behavior is associated with worse prognosis for anorectic individuals. The BN and ANP groups had the same common 3-point scale code elevation of scales 2, 4, and 8, with scale 4 being the peak. The ANR had a 3-point code of scales 2, 8, and 7, with scale 2 being the peak. Results indicate that binge/purge behavior and restricting behavior may be expressed in different scale elevations.

In a review of Casper, Hedeker, and McClough's 1992 study, Cumella et al. (2000) considers their results comparing MMPI profiles of individuals diagnosed with ANR, ANP, and BN. Consistent with other research, ANR profiles were found to be consistently in the subclinical range, or elevated solely on scale 2. In contrast, mean MMPI profiles for ANP and BN were elevated on 3-6 scales. The most common 3-point elevation for ANP and BN was scales 2, 4, and 8.

Scott and Baroffio (1986) compared MMPI profiles of individuals diagnosed with AN, BN, morbid obesity, and a control group. The study found that overall the three disordered eating groups had considerable similarities in overall profiles, but they were significantly different than the control group. The AN and BN were considerably more elevated than the morbidly obese on Scale 8, which Scott and Baroffio (1986) suggest could indicate that they have greater identity confusion and reality distortion. In contrast to previous studies, the AN group had a more elevated profile. This could be due to the combining of ANR and ANP. Previous research has shown when the AN group is split into ANR and ANP you can see an elevated profile with ANP that you do not see with ANR.

Despite the MMPI's widespread use, critics expressed many valid concerns about the instrument, pertaining to the limits of the original standardization sample, archaic item content, inappropriate language, and missing representation of psychopathology content due to the empirical manner in which the scales were constructed, the interscale correlation, and item overlap is high between the clinical scales (Graham, 2011). The criticisms led to a revision of the original instrument.

MMPI-2 and Disordered Eating

The MMPI was revised and published as the MMPI-2 in 1989 (Butcher, Dahlstrom, Graham, Tellegen, & Kaemmer, 1989). The primary goals for the revision of the MMPI included collecting a contemporary normative sample that would be representative of the general population and improving the item pool by removing outdated material and generating new items that would expand the content dimensions. Despite some previous criticisms of the outdated MMPI scales, it was determined that all efforts would be made to maintain continuity between the MMPI and the MMPI-2 so as not to lose the considerable research base and professional following that had accumulated with the MMPI. The new normative sample consisted of 2,600 adults from across the country that better represented the U.S. population. The MMPI-2 consisted of 567 items and was comprised of the same Clinical scales as the original MMPI. A t-score above a 65 on any scale in the MMPI-2 is considered to be clinically significant (Graham, 2011). Because the MMPI-2 used a more representative sample and more carefully derived content scales with additional content, the studies using the MMPI-2 to determine scale elevation differences between eating disorder types could potentially be more accurate than studies previously conducted with the MMPI. The following is a review of those findings.

Pryor and Weideman (1996) compared MMPI-2 profiles of women meeting DSM-IV diagnostic criteria for ANR, ANP, BNP, or BNPNP. They found they could not distinguish between the Clinical scale elevations for any of the four groups. Elevations on scales 2 and 7 were most common for all groups, indicative of depression and emotional distress. In contrast to results from much of the previous research using the MMPI, ANR had more elevations than previously seen with the first iteration of the instrument, and ANP had fewer elevations, with scale 2 being the only primary elevation. Common elevations of scales 4 and 6 were seen for BN participants, indicative of impulsivity and lack of constraint, compared to scales 4 and 8

commonly elevated with the MMPI. The study was later criticized for a very small sample size and resulting low power. Further, the population in the study was outpatient individuals, who are often less severe than residential or hospitalized patients (Cumella et al., 2000).

Cumella et al. (2000) compared MMPI-2 profiles of women diagnostically categorized as ANP, ANR, BN, or EDNOS. Similar to previous research, they found the most common 2-point Clinical scale elevation code in all four groups was 2-7. The 2-7/7-2 code is characterized by anxious and depressive symptoms, including low self-esteem, worry, guilt, rumination about perceived personal inadequacies, and possible obsessive-compulsive behaviors. While the groups were all inclusively similar, they were two to three times more likely to obtain a 2-7/7-2 code than other psychiatric patients. These findings suggest the possibility of a meaningful relationship between the personality characteristics of the 2-7/7-2 profiles and the development or maintenance of the several eating disorders. Similarly, they also found that the most common 3-point Clinical scale elevation occurred for all groups in the same order of scales 2, 7, 3 characterized as a docile, passive individual with strong interpersonal dependency issues who may be adept at inspiring others to take care of them. Beyond the 3-point scale elevations, all four groups were elevated on the same 6 scales: 1, 2, 3, 4, 7, and 8.

This study did find some differentiation with ANR. The validity scales were able to establish that ANR individuals consistently reported less psychopathology than other groups in the study. This group also had a tendency for lower scores on scale 8.

Exterkate, Bakker-Brehm, and Jong (2007) examined the MMPI-2 profiles of women diagnosed with ANR, ANB, BNP, BNN, and EDNOS. Similar to Cumella et al. (2000) and Pryor and Weiderman (1996), they found the most common 2-point code to be 2/7. Again the

only differences were found in ANR group, where scales 1 and 2 were significantly lower in ANR than in ANP.

The second iteration of the MMPI instrument may emphasize the similarities between eating disorder diagnostic groups more than the differences. These studies were completed with an older version of the MMPI that has been criticized for high interscale correlation and over-inclusive measures of demoralization that may have contributed to the inability to consistently differentiate between disordered eating behavior.

MMPI-2-RF and Disordered Eating

Tellegen et al. (2003) developed the Restructured Clinical scales, due to criticisms of over-inclusive item content, high interscale correlation, and item overlap in the MMPI-2 Clinical scales. Consensus identified that the construct of demoralization permeated through all Clinical scales; consequently, distress and emotional turmoil were effectively causing over inflation of scores on all Clinical scales. One of the primary goals of developing the Restructured Clinical scales was to isolate the measure of demoralization to more effectively measure the core constructs of the Clinical scales, and consequently improve discriminant validity of the Clinical scales (Ben-Porath, 2012). The nine RC Scales are as follows: RCd (demoralization), RC1 (somatic complaints), RC2 (low positive emotion), RC3 (cynicism), RC 4 (antisocial behavior), RC6 (ideas of persecution), RC7 (dysfunctional negative emotions), RC8 (aberrant experiences), and RC9 (hypomanic activation) (Tellegan et al., 2003). Elevated RCd scores are indicative of distress, emotional turmoil, unhappiness, and dissatisfaction. Elevated RC1 scores are indicative of specific somatic complaints, neurological, gastrointestinal, or general pain-related complaints. Elevated RC2 scores are indicative of low positive emotions, anhedonia, and depression-related symptoms. Elevated RC3 scores are indicative of cynical beliefs, distrustfulness, and beliefs that

others look out for their own interests. Elevated RC4 scores are indicative of antisocial behavior, including criminal history, impulsivity, failure to conform to societal norms, and family problems. Elevated RC6 scores are indicative of ideas of persecution, experiences of paranoid delusional thinking, and suspiciousness and alienation from others. Elevated RC 7 scores are indicative of dysfunctional negative emotions, including anxiety, intrusive ideation, anger proneness, and fear. Elevated RC8 scores are indicative of unusual dysfunctional thought patterns and perceptions, and unrealistic thinking. Elevated RC9 scores are indicative of hypomanic activation, including restlessness, aggression, mood instability, and excitability. Scale T scores above a 65 are considered to be clinically significant.

Using these scales, Ben-Porath and Tellegen developed the MMPI-2-Restructured Form, focusing on a dimensional approach to measuring personality and psychopathology (2008/2011). The Restructured Form contains 338 items that accommodate a total of 51 scales: 9 validity scales and 42 substantive scales organized in a hierarchical fashion. The three higher order scales include measurements of emotional/internalizing dysfunction (EID), thought dysfunction (THD), and behavioral/externalizing dysfunction (BXD). The validity scales include measures of overreporting, underreporting, and unscorable responses, including Cannot Say (CNS), Variable Response Inconsistency (VRIN-r), True Response Inconsistency (TRIN-r), Infrequent Responses (F-r), Infrequent Pathology Responses (FP-r), Infrequent Somatic Responses (Fs), Symptom Validity (FBS-r), Response Bias Scale (RBS), Uncommon Virtues (L-r), and Adjustment Validity (K-r). Since its creation, the MMPI-2-RF has proven to be a reliable and valid measure of personality and psychopathology (Ben-Porath & Tellegen, 2008/2011).

After extensive review of literature, there appear to have been no published studies using the MMPI-2-RF Restructured Clinical scales to differentiate disordered eating behavior.

However, there have been a few unpublished dissertations examining the relationship. Previous research has shown the MMPI and MMPI-2 provided results that indicated DSM diagnosed eating disorders had more scale similarities than differences; however, considering the previously discussed criticisms of the first two iterations of the instrument, further research needs to be done that utilize the revised instrument and examines disordered eating in a dimensional, non-diagnostic fashion.

Electroencephalography

Electroencephalography (EEG) is the recording of electrical activity in the cortex through one or more electrodes attached to the scalp. The records typically reflect the synchronized and desynchronized oscillations of the overall cortical activity in the brain. Quantitative EEGs express these electrical variations in numbers versus just patterns of wave forms. Some EEGs can localize the areas within the brain where specific activity is occurring. EEG is a useful tool for identifying electrical abnormalities in the cortex.

Brain waves are historically categorized into four primary groups delineated by frequency: delta: 0.5–4 hertz (Hz); theta: 4–8 Hz; alpha: 8–13 Hz; beta: >13 Hz (Jáuregui-Lobera, 2012). Brain electrical activity creates complex wave shapes. Within these complex forms there are component waves that are commonly sinusoidal. The brain state of the individual can be described with specific measures of the component frequencies. For example, the waves' amplitude and frequency may be combined as an expression of "power." Few waves with low amplitude would be showing little power. Many waves with high amplitude would show maximum power. An EEG with many alpha waves is considered a state of "relaxed wakefulness," and beta waves suggest a state of "alert attentiveness." Cortical activation would be represented by low alpha power and increased beta power (Hatch et al., 2011).

Previous research has found that brainwave abnormalities may be related to and aid in identification of certain pathologies. For example, global EEG can identify abnormal connectivity and the ratio of theta and beta waves has been related to Attention Deficit/Hyperactive Disorder (Alba et al., 2016; Gonzalez et al., 2013). A significantly higher power in theta and beta bands enabled researchers to differentiate between a depressive state in individuals with major depressive disorder and individuals with bipolar disorder (Woźniak-Kwaśniewska, Szekely, Harquel, Bougerol & David, 2015). Decreased theta, alpha, and beta activity has been associated with generalized anxiety disorder (Demerdzieva, 2011).

Electroencephalography and Disordered Eating

Research looking at EEG correlates of eating disorders has been limited. Most previous research has focused primarily on anorexia. Other attempts have been made to explore EEG correlates with compulsive eaters, chronic dieters, prognostic prediction, and typology of disordered eating behaviors; however, to date, these studies have not been consistent or conclusive (Jáuregui-Lobera, 2012).

Jáuregui-Lobera (2012) found that before and after weight restoration individuals with anorexia showed reduced relative alpha power and increased relative beta power during the ‘eyes open’ EEG. However, a significant increase in alpha power and decrease in beta and delta power was observed in the ‘eyes closed’ EEG in the participants that were measured again after weight restoration. Additionally, a recent study specifically looking at the impact of refeeding on adolescent anorexia EEG correlates, also found that in “‘eyes open,’” underweight participants exhibit reduced relative alpha power and increased relative beta power. In the “‘eyes closed’” period, underweight participants had elevated theta in parietal-occipital regions, a trend that remained after refeeding (Hatch et al., 2011). In contrast, the only lasting effect after weight

restoration in this study was the increased theta power, while alpha and beta abnormalities were not significant after refeeding. Evidence of lasting abnormalities would support the possibility of potential irreversible damage after prolonged period of starvation or a “scar tissue” effect for an undetermined period of time after weight restoration. In contrast, in several other studies similar abnormalities have been found in underweight individuals that have completely normalized with refeeding (Jáuregui-Lobera, 2012).

The debate highlights whether the reported EEG abnormalities are an effect of structural brain changes related to starvation or are predisposing disease markers of anorexia nervosa (Jáuregui-Lobera, 2012; Hatch et al., 2011). Different studies have reported cortical dysfunctions in patients with anorexia nervosa even after weight gain, whereas others have reported a normalization of EEG in those patients who have weight restored. The controversy has been difficult to resolve due to the variability in several studies’ samples in terms of age, body mass index, EEG technique used, measurement of illness-duration, and experimental settings (Jáuregui-Lobera, 2012; Hatch et al., 2011). Generally, findings have been inconsistent and further research needs to be done to clarify the apparent trends with EEG activity and disordered eating behaviors. Brain changes in bulimia nervosa seem to be less pronounced than in anorexia nervosa and are mainly due to chronic dietary restrictions. Different subtypes of eating disorders might be correlated with specific brain functional changes (Jáuregui-Lobera, 2011).

Statement of Problem

Considering the mortality rates and low treatment efficacy, eating disorders are diagnoses that should receive serious attention by mental health professionals and researchers. Previous research suggests that personality differences among eating disorders may be predictive of symptomatology, treatment response, and prognosis (Cassin & Von Ranson, 2005). Previous

studies have not been published using the MMPI-2-RF, which is presumably more valid than earlier forms. Additionally, the association of eating disorder symptomology and cortical abnormalities has been explored with inconsistent results. Studies regarding EEG correlates and eating disorders have been limited due to sample size, combined eating disorder behaviors, and heterogeneous sample populations. Following the RDoC framework, this study aims to use the MMPI-2-RF to look at personality and psychopathology differences between a dimensional array of disordered eating behaviors spanning restricting, purging, and binge-eating behaviors, along with a physiological measure of 5-point EEG data to examine associated brain wave patterns.

Hypotheses

Hypothesis 1: It is hypothesized that measures of constructs related to internalizing dysfunction, including scales EID, RCd, RC2, RC7, and EEG abnormalities, including decreased low band and increased high band, will be significant predictors of engaging in restrictive behaviors, including dieting and fasting.

Hypothesis 2: It is hypothesized that measures of constructs related to externalizing dysfunction, including scales BXD, RC4, RC9, will be significant predictors of engaging in purging behaviors, including vomiting, enema use, diuretic use or laxative use.

Hypothesis 3: Externalizing RC scales BXD, RC4, and RC9 should be significant predictors for binge-eating behavior for the non-clinical population.

Hypothesis 4: Internalizing RC scales EID, RCd, RC2, and RC7 should be significant predictors for binge-eating behavior for the clinical population.

CHAPTER 3: METHODOLOGY

Participants

Participants forming the non-clinical population were 37 adult individuals enrolled at Western Carolina University undergraduate program participating in partial fulfillment of a course requirement. Five participants had to be discarded due to four data sets with invalid MMPI-2-RF validity scales and one incomplete data set, leaving 32 remaining participants. Included participant ages ranged from 18 to 30 years old with an average of 20.91 years. 43.75% of the participants were male and 56.25% were female. Research suggests traditional college students are at increased risk for eating disorder development (Hudson et al., 2012) and increased non-clinical disordered eating (Lundahl, Wahlstrom, Christ et al., 2015); therefore, the non-clinical participants were thought to provide a healthy control group, but were expected to reveal a moderate range of disordered eating behaviors.

Participants forming the clinical population were six adult female individuals admitting to a CARF Accredited, state-licensed, residential eating disorder treatment facility in western North Carolina. Participant ages ranged from 18 to 27 years old with an average of 20.0 years. Admitting clients had been clinically diagnosed with eating disorders fitting DSM-5 criteria, including anorexia nervosa, bulimia nervosa, binge-eating disorder, or other specified/unspecified feeding and eating disorders. Disorder severity was controlled for by insurance utilization review standards for residential treatment authorization criteria, as well as, admission severity limits placed by the facility, including a body mass index minimum requirement of 14, no active psychotic symptoms, no actively suicidal plans, and/or the client cannot be actively detoxing from any substance. Upon admission, clients were asked if they

would like to participate in research to investigate psychopathology and EEG correlates of disordered eating to improve diagnostic means. Clients were free to decline participation; 42.9% of admitting clients during the data collection period consented to participation. The data were collected within 48 hours of admission to the treatment center to minimize psychological and physiological effects of treatment. Exclusion criteria for both populations were standard for the MMPI-2-RF: VRIN \geq 80; TRIN \geq 80; CNS \geq 15; F-r \geq 120; and Fp-r \geq 100.

Measures

Personality and psychopathology were measured by the Minnesota Multiphasic Personality Inventory-2-Restructured Form, a 338-true/false-item assessment of 51 scales, including 9 validity scales, 3 higher order scales, and 39 substantive scales. The reliability and validity information was adhered per the MMPI-2-RF Technical Manual (Ben-Porath & Tellegen, 2008/2011).

A quantitative EEG was performed on each participant for 5 minutes eyes open and 10 minutes eyes closed. Levels of low band power, high band power, delta power, and theta power were obtained with measures of amplitude and frequency for left and right hemispheres. This study used a 5-point iWorks EEG machine and the LabScribe system to display and analyze results.

A disordered eating questionnaire was completed through a Qualtrics survey. The disordered eating questionnaire was created by breaking down the DSM-5 diagnostic criteria for anorexia, bulimia, and binge-eating disorder. By breaking down all of the diagnostic symptoms it enables dimensionalization of the behaviors to explore correlates to specific behaviors without arbitrary diagnostic cut offs. For each disordered eating behavior, a question is presented on a 7-

point scale pertaining to frequency, denoting “never,” “occasionally,” “monthly,” “a few times per month,” “weekly,” “several times per week,” or “daily.” (See Figure 1)

Procedure

Participants completed the on-screen administration of the MMPI-2-RF in a computer room with minimal noise and distraction, taking approximately 35-50 minutes. Next, they took the disordered eating questionnaire in the form of a Qualtrics survey, taking approximately 10 minutes. Lastly, they received a 5-point EEG from a trained individual in an environment that was quiet and private. The EEG consisted of a 5-minute recording with the participant’s eyes open facing a wall with no stimulus, followed by a 10-minute recording with their eyes closed with instructions to “relax as much as you can.” The procedure took approximately 60-75 minutes per participant.

CHAPTER 4: RESULTS

To investigate the first hypothesis, a stepwise multiple logistic regression was run using the MMPI-2-RF broad EID scale and RC scales RCd, RC2, RC7 as predictors to determine which linear combination best predicts restricting behavior in a non-clinical population, specifically dieting or fasting, and to determine the accuracy of the classification. Due to extensive anomalies found throughout the EEG data, presumably due to technological error, the stepwise multiple logistic regression was not conducted using the EEG measures of decreased low band and increased high band as predictors as originally hypothesized. The multiple regression analyses found no significant results using the MMPI-2-RF internalizing scales EID, RCd, RC2, or RC7 as predictors of the restricting behaviors dieting or fasting.

To investigate the second hypothesis, a stepwise multiple logistic regression was run using the MMPI-2-RF broad BXD scale and RC scales RC4 and RC9 as predictors to determine which linear combination best predicts purging behavior in a non-clinical population, specifically vomiting, diuretic use, or laxative use, and to determine the accuracy of the classification. No participants reported using enemas, so the multiple regression analyses could not be executed for this purging behavior as hypothesized. Of the predictors variables, only the RC4 scale (Antisocial Behavior) was found to predict vomiting ($\beta = .413$; $t(31) = 2.484$; $p = .019$), accounting for 17.1% of the variability in the dependent variable. Since only one of 32 subjects reported using both diuretics and laxatives, the multiple regression analyses for these dependent variables found nothing of significance.

To investigate the third hypothesis, a stepwise multiple logistic regression was run using the MMPI-2-RF broad BXD scale and RC scales RC4 and RC9 as predictors to determine which

linear combination best predicts binge-eating behavior in the non-clinical population, and to determine the accuracy of the classification. Of the predictors variables, only the RC9 scale (Hypomanic Activation) was found to predict binge-eating behavior ($\beta=.413$; $t(31) = 2.483$; $p = .019$), accounting for 17% of the variability in the dependent variable.

To investigate the fourth hypothesis, a stepwise multiple logistic regression was intended to be run using RC scales RCd, RC2, and RC7 as predictors to determine which linear combination best predicts binge-eating behavior in the clinical population, and to determine the accuracy of the classification. However, due to a clinical population sample size of only six participants, no regression analyses were able to be conducted to investigate this hypothesis.

For all analyses, due to the size of the sample, the full set of multiple regression diagnostic procedures such as distance, leverage, and influence could not be calculated. Typically, these diagnostic procedures require a sample size of at least 300 subjects to be conducted with any degree of accuracy. Additionally, the small sample size did not allow for cross-validation between clinical and non-clinical samples of the regression models with significant predictors (Howell, 2013).

While regression analyses were not able to be conducted on the six clinical population participants due to small sample size, some descriptive information regarding disordered eating behavior endorsement was obtained and presented in Table 1 in the Appendices. Indicative of the clinical nature of the population, 100% of participants endorsed feeling the negative consequences of eating to some degree, experiencing a marked eating-related interference with their social lives, and intense fear of gaining weight or becoming fat.

CHAPTER 5: DISCUSSION

While the data are preliminary, two significant MMPI-2-RF scale predictors of specific disordered eating behaviors were found. The RC4 scale (Antisocial Behavior) is positively and significantly correlated with the disordered eating behavior of purging by vomiting, indicating that those with higher scores on the scale indicative of antisocial behavior, impulsivity, criminal history, failure to conform to societal norms, and family problems tend to report higher frequency of purging by vomiting in the non-clinical student population. This finding is consistent with much of the previous research regarding 2-point and 3-point scale elevations containing scale 4 being associated with both anorexia nervosa binge/purge type and bulimia nervosa (Cumella et al., 2000; Norman & Herzog, 1983; Pryor and Weideman, 1996). While bulimia nervosa indicates compensatory behaviors other than vomiting, research indicates that as much as 70-80% of individuals diagnosed with bulimia nervosa purge by means of vomiting (Goldsmith, 2016). This study's results are consistent with previous research and indicate that RC4 is predictive of the behavior of purging by vomiting specifically.

Secondly, the RC9 scale (Hypomanic Activation) is positively and significantly correlated with the disordered eating behavior of binge-eating, indicating that those with higher scores on the scale indicative of hypomanic activation, restlessness, aggression, mood instability, and excitability tend to report higher frequency of binge-eating in the non-clinical student population. After extensive literature review, this scale correlation has not been found in previous literature; however, a similar association by bivariate correlation was found in the pilot study proceeding this project with a non-clinical student population acquired by the same selection process. Due to small sample size and selection criteria, this result is not at this time

generalizable to the general population, but may be reflective of the population attained for this study and pilot study or a finding that is relative to non-clinical disordered eating. Future data is needed regarding this association.

This study had several limitations that may have affected the results. Firstly, extensive anomalies were found throughout the EEG data that was consequently discarded; the errors are thought to have been due to technological dysfunction of EEG leads or other equipment. Future research would benefit from accurately attained brain wave measures that provides clarity to the inconsistent research regarding brain wave abnormalities in individuals with disordered eating. Secondly, non-clinical student participants were only gathered from a small, rural university in western North Carolina. This population may have notable differences from the general population that may affect the generalizability of these results. Thirdly, a very small sample size of only six participants for the clinical population is not large enough to perform predictive or correlational statistics or to generalize to the broader eating disorder population. Smaller participant numbers than expected resulted from a higher than anticipated rate of declining participation of 57.14% and a slower than expected admission rate per the facility. Future research may benefit from both a non-clinical and clinical sample of appropriate size to make further conclusions regarding the relationships between psychopathology/personality scales of the MMPI-2-RF, EEG brain wave activity, and dimensional disordered eating behavior in a clinical and non-clinical population.

Considering the substantial rates of comorbidity among eating disorders, research advances in the relationship between psychopathology and eating disorders could increase the diagnostic specificity and accuracy of eating disorders. Additionally, while substantial future research would need to be conducted, specific psychopathology scales significantly predicting

individual eating behaviors suggests that different treatment methods for specific behaviors may best address their overall symptomology. This specific treatment would replace treatment methods based on arbitrary, categorical diagnoses, in which there is significant diagnostic crossover (American Psychiatric Association, 2013; Eddy et al., 2008; Milos et al., 2005; Tozzi et al., 2005). Further, considering the prevalence of comorbidity, clients should be evaluated for comorbid antisocial behavior such criminal history, impulsivity, failure to conform to societal norms, and family problems when being treated for purging by vomiting behavior. Treatment should include a focus on these other problems related to their behavioral dysfunction. This may lead to the best treatment outcomes overall.

While this study had several limitations, some of the results found are promising. MMPI-2-RF scale elevations predictive of specific disordered eating behaviors advances the understanding between psychopathology and eating disorders that could result in improved diagnostic specificity and accuracy of eating disorders. Taking a dimensional approach to assessing psychopathy, disordered eating behaviors, and physiological measures may improve diagnosis and subsequent treatment methods for individuals with eating disorders. Considering the substantial comorbidity and unmatched mortality rates, any improved understanding, diagnostic specificity, and information to guide future treatment research provides needed progress.

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APPENDIX

Table 1
Clinical participants' disordered eating behavior endorsement

(n=6)	Never	Some level of endorsement	Daily
Restricting of intake relative to dieting	0	3	3
Restricting of intake relative to fasting	1	5	0
Restricting of intake relative to excessive exercise	3	2	1
Binge-eating: eating, in a discrete period of time (e.g., 2-hour period), an amount of food that is definitely larger than what most individuals would eat in a similar period of time under similar circumstances.	2	3	1
Purging: Vomiting	1	4	1
Purging: Laxatives	4	1	1
Purging: Diuretics	6	0	0
Purging: Enemas	5	1	0
Concern about aversive consequences of eating	0	3	3
	No	Yes	
Food avoidance based on sensory characteristics of food	4	2	
Significant weight loss (or failure to achieve normal weight) related to eating behavior	2	4	
Significant weight gain related to eating behavior	4	2	
Significant nutritional deficiency related to eating behavior	3	3	
Eating-related marked interference with your social life	0	6	
Intense fear of gaining weight or becoming fat	0	6	

Figure 1. *Eating Disorder Symptom Questionnaire*

	Never	Occasion	Monthly	A few times/ month	Weekly	Several times/ week	Daily
Restricting of intake relative to dieting	1	2	3	4	5	6	7
Restricting of intake relative to fasting	1	2	3	4	5	6	7
Restricting of intake relative to excessive exercise	1	2	3	4	5	6	7
Binge-eating: eating, in a discrete period an amount of food that is larger than what most individuals would eat in a similar period of time/circumstances.	1	2	3	4	5	6	7
Purging: Vomiting	1	2	3	4	5	6	7
Purging: Laxatives	1	2	3	4	5	6	7
Purging: Diuretics	1	2	3	4	5	6	7
Purging: Enemas	1	2	3	4	5	6	7
	Yes	No					
Food avoidance based on sensory characteristics of food	1	2					
Concern about aversive consequences of eating	1	2					
Significant weight loss (or failure to achieve normal weight) related to eating behavior	1	2					
Significant weight gain related to eating behavior	1	2					
Significant nutritional deficiency related to eating behavior	1	2					
Eating-related marked interference with your social life	1	2					
Intense fear of gaining weight or becoming fat	1	2					